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EFFECT OF THE ENVIRONMENT ON ASTHMA IN CHILDREN.

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INTRODUCTION

"Rates of hospital admission/separation for asthma and rates of death from asthma have increased dramatically among children and young adults during recent years in Canada."(1). In the United States, asthma is the most common chronic disease of childhood, affecting 15 percent of Americans under the age of 15. "It ranks first among the chronic diseases in causing school absenteeism, and it has been linked to lowered academic performance."(2) Asthma is the commonest cause for admission to hospital in North America and twice as common as the next most common cause (6). The rate of hospitalization in the U.S. for children under 15 years old with asthma has increased at least 145% in the period 1970 to 1984 (12). Studies have also shown that asthma often continues into adult life, with only brief improvement in adolescence (3,4). Appropriate diagnosis and treatment reduces morbidity in childhood, and it is possible that good control in childhood may reduce severity in adult life (3,5).

These are disturbing statistics, both from the point of view of public health, and from the point of view of resource costs in the health care system. It is not clear whether the increase in morbidity and mortality is a result of increasing prevalence or severity, or by past underdiagnosis and undertreatment, or by a combination of factors. However, there is some evidence in the literature that the respiratory environment plays a role in the pathogenesis of asthma.

Environmental factors associated with asthma/wheeze.

Becklake et al. were not able to demonstrate any effect of air pollution on the prevalence of asthma in their study in Montreal (10). Weiss et al. found that persistent wheeze was associated with parental smoking, in their study of children in the Boston area. Environmental factors examined included the use of gas heaters in the kitchen, and crowding in the home, neither of which were found to be associated with persistent wheeze (9). Schenker et al. were not able to confirm the observation of Weiss and co-workers that persistent wheeze was associated with parental smoking. In their study, in rural Pennsylvania, they also included socioeconomic status (SES) and the use of gas stoves for cooking. Lower SES was found to be associated both with the use of gas stoves and increased prevalence of persistent wheeze, but when SES was accounted for, no effect of gas stove use on prevalence of persistent wheeze was found (7). Ware and co-workers, in the "Six Cities" study, found strong effects of parental smoking on "wheeze" in their study of over 10,000 children (13). They did not observe an effect of gas stove use on the prevalence of wheeze, and in a later report were not able to observe an effect of air pollution on "wheeze" although significant associations with increased prevalence of lower respiratory illness were observed (8). Fergusson et al. were not able to show an association of asthma with parental smoking, but did observe that parental smoking contributed to increased rates of lower respiratory infection in the first two years of life. A number of other environmental factors (socioeconomic, pets in the home) were examined, and were not reported to be associated with increased asthma prevalence (11). Silverman and co-workers reported acute effects of suspended particulates on asthmatic adult subjects which changed in direction depending on the season, and found difficulty in interpreting these data (17). Thus there is little evidence to date that air pollution has an effect on either the prevalence or the severity of asthma in children.

Background.

We have been examining the effect of environmental factors on the respiratory health of a cohort of over 3200 schoolchildren in Hamilton, Ontario. Design, methods, and some early results have been previously reported (14,15,16). This communication reports on an aspect of the study which has only recently arisen from a specific analysis of asthmatic children found in this cohort.

METHODS

A stratified random sample of over 3200 children was drawn from all children born after 1968 attending Levels (corresponding to 'Grades') 2, 3, and 4 of the schools of the Board of Education of the City of Hamilton in September 1978. The overall design was to use respiratory health as an outcome or dependent variable. Respiratory health was assessed in part from pulmonary function measurements made in the schools, and in part on the basis of parents' responses to questions about the health of the child in the previous year. The major independent variable of concern was air pollution, but important covariables included the child's respiratory history as an infant, as well as other environmental covariables, such as parental smoking and the use of gas for cooking. The objective was to determine the relative importance of the different independent variables in determining the state of respiratory health at the time of measurement.

Parents (usually the mother) were asked questions relative to the child's home environment, in addition to questions on respiratory health. These questions provided information on housing, socioeconomic status, health of family members other than the study child, cooking fuel, and the smoking habits of the household.

Pulmonary function testing was performed in the school. The apparatus used was an Hewlett-Packard pneumotachograph based Pulmonary Calculator System. Computations were performed internally and a graphic output of records was obtained for each child at the time of testing. Measurements were made of indicators derived from the forced vital capacity manoeuvre, as well as the single- and multiple-breath nitrogen washout manoeuvre. For the purposes of this report, two indicators derived from the FVC will be used to describe pulmonary function: FEV_1/FVC (Ratio of volume forcibly exhaled in the first second of a maximum exhalation, to the forced vital capacity); and MET (Mid-Expiratory Time, the period during which the middle half of the forced vital capacity is exhaled).

Exposure to SO_2 was determined using data obtained from a network of 10 continuous SO2 analysers, and exposure to the fine fraction (FF; <3.3 um AMMD) of suspended particles from a network of 7 Andersen cascade impactors. Spatial gradients in SO_2 and FF were found to exist over the city, and 1 year mean exposures were calculated for each child according to the geographical location of their school. The method of calculation of exposure depended on the generation of a 3-dimensional 'surface' based on monthly mean data, which represented the pollutant concentration in the 'z' axis, as a function of the geographical 'easting' and 'northing' coordinates in the 'x' and 'y' axes respectively. From this surface, the exposure of a child to the pollutant was obtained by estimating its concentration from the surface at the location of the school the child attends. A 1-year exposure was obtained by determining the arithmetic mean of 12, monthly mean, school-based values.

For each pollutant, the children were divided into two groups: those with exposure above the median level (for SO_2, 10.6ppb; for FF, 46 ug/m^3), and those below. Each group was further divided into children with a physician diagnosis of asthma, and those

without. Comparison of the value of the two pulmonary function variables FEV_1/FVC and MET was made between groups. Data presented here are for what we refer to as "Period 2", corresponding essentially to the calendar year 1980, and the mean age of the cohort at this time was 10 yr.

RESULTS

We analysed the response of the asthmatic children to both SO_2 and FF in Period 2, compared to the rest of the cohort. Results are shown in the Table on the next page. From this Table it may be seen that there were no statistically significant changes in airflow obstruction in asthmatic children associated with a higher level of exposure to fine fraction, but there were statistically significant but clinically unimportant decreases in airflow obstruction in normal children associated with a higher exposure to fine fraction, detected only in MET but not in FEV_1/FVC.

We found statistically and clinically significant increases in airflow obstruction in the asthmatic group demonstrated by two reliable indicators of pulmonary function, associated with increased chronic exposure to SO_2 at the relatively low levels found in Hamilton during this period (median 10.6 ppb, range 8.4-17.5 ppb annual arithmetic mean). This was not observed in the rest of the cohort; in fact statistically but not clinically significant decreases in airflow obstruction were observed in the rest of the group, consistent with the earlier observations with respect to SO_2 we reported in November 1986. The former is a particularly surprising finding, because it demonstrates airflow obstruction in asthmatic children associated with SO_2 exposure levels one to two orders of magnitude less than previously observed for acute exposures in adults.

CROSSECTIONAL ANALYSES OF PULMONARY FUNCTION OF NORMAL AND ASTHMATIC CHILDREN
EXPOSURE TO FINE FRACTION
(Median 46; min 34; max 59 ug/m^3)

PULMONARY FUNCTION	MALES		FEMALES		COMBINED	
	ASTHMA	NORMAL	ASTHMA	NORMAL	ASTHMA	NORMAL
(NUMBER)	(68)	(1564)	(46)	(1493)	(114)	(3057)
FEV1/FVC						
FF ABOVE	0.742	0.796	0.754	0.828	0.746	0.812
FF BELOW	0.764	0.801	0.780	0.829	0.771	0.814
MET				*		*
FF ABOVE	0.791	0.617	0.808	0.538	0.798	0.577
FF BELOW	0.744	0.622	0.721	0.554	0.734	0.590

EXPOSURE TO SULPHUR DIOXIDE
(Median 10.6; min 8.4; max 17.5 ppb)

PULMONARY FUNCTION	MALES		FEMALES		COMBINED	
	ASTHMA	NORMAL	ASTHMA	NORMAL	ASTHMA	NORMAL
(NUMBER)	(68)	(1564)	(46)	(1493)	(114)	(3057)
FEV1/FVC					*	
SO2 ABOVE	0.728	0.797	0.754	0.830	0.737	0.813
SO2 BELOW	0.775	0.800	0.776	0.827	0.776	0.813
MET	*			*	*	*
SO2 ABOVE	0.842	0.618	0.822	0.537	0.835	0.578
SO2 BELOW	0.702	0.622	0.725	0.556	0.712	0.590

Notes:
Decrease in FEV1/FVC reflects increase in airflow obstruction.
Increase in MET reflects increase in airflow obstruction.

Significance: $p < 0.05$ = (*)

DISCUSSION

Increased MET indicates increased airway obstruction, as does a decreased FEV_1/FVC ratio, and children with asthma show both increased MET and decreased FEV_1/FVC compared with normal children. Children with asthma show a further clinically significant increase in MET and decreased FEV_1/FVC associated with increased SO_2 levels, while normal children show a clinically not significant reduction in MET only, with increased SO_2. These observations suggest that 1-year exposure to increased SO_2 in the 10-20 ppb range is associated with an increase in airway obstruction in asthmatic children, not observed in normal children.

We believe that our observations are of major importance, when viewed in the context of the "acid rain" problem. SO_2 is likely to be a component of "acid rain" which has a substantial impact on respiratory health, but the levels associated with "long-range transport" typically are well below the levels where acute effects have been shown on asthmatic subjects in controlled environmental chamber studies. Demonstration of clinically significant effects at low levels in a sensitive population underline the importance of better SO_2 control.

Asthma as a New Public Health Concern.

The recognition of an increase in the prevalence, morbidity, and mortality of asthma has been well documented recently in many industrialized countries, and whether there is a causal link to "acid rain" has not been established. In our own study, asthma prevalence was not found to be associated with higher levels of SO_2; but we have demonstrated that the consequences of chronic SO_2 exposure to asthmatic subjects are to further decrease their already compromised pulmonary function.